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Risk and Resilience Factors in the Etiology of Chronic Posttraumatic Stress Disorder

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As noted several places in this volume, a number of important large-scale studies have documented that experiencing a traumatic event is not uncommon in our modern society (e.g., Breslau, Davis, Andreski, & Peterson, 1991; Davidson, Hughes, Blazer, & George, 1991; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Norris, 1992; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). Moreover, posttraumatic stress disorder (PTSD) is a significant mental health problem, with U.S. prevalence estimates of lifetime PTSD approximating 8%, more than 10% for women, and about 5% for men (Kessler et al., 1995). On the other hand, the preponderance of trauma victims—though possibly quite symptomatic in the early postexposure period—tend to recover and adjust with the passage of time. Therefore, other factors must contribute to the likelihood that exposure to traumatic events and circumstances will have long-term mental health consequences. These factors may include features of the traumatic event itself, preexisting demographic and other individ-

ual differences characteristics of the trauma victim, and aspects of the post-exposure recovery environment, among others. Collectively, these are often referred to as *risk and resilience factors.* Other terms that are commonly used are *vulnerability or hazard and protective or preventive factors,* respectively.

It is important at the outset to clarify the terms "risk factor" and "resilience factor." In the context of negative mental health consequences of trauma exposure, we use the term "risk factor" to label those characteristics of the event, the individual, or the environment that are associated with an increase in PTSD. We use the term "resilience factor" to label those characteristics of the event, the individual, or the environment that are associated with a decrease in PTSD. We view this as simply a linguistic convenience because, in effect, the opposite pole of a risk factor (e.g., low socioeconomic status) could be judged a resilience factor (e.g., high socioeconomic status). Likewise, the opposite pole of a resilience factor (e.g., the presence of strong social support in the recovery environment) could be judged a risk factor (e.g., the absence of a structural or functional social support system following the trauma).

In this chapter, we seek to integrate substantive evidence concerning risk and resilience for PTSD with the special methodological issues that must be confronted in understanding the etiology of PTSD. In the first section to follow, we provide a brief overview of factors that have been shown to covary, either positively or negatively, with PTSD symptomatology. In the next section, we propose four methodological recommendations to assist researchers to validly assert the place of particular risk and resilience factors in the network of causal associations leading to PTSD. We close with a summary commentary on implications for prevention, treatment, and policy.

OVERVIEW OF RISK AND RESILIENCE LITERATURE

The recognition that PTSD is by no means a universal response to trauma has inspired a growing body of literature aimed at documenting risk and resilience factors for PTSD following trauma exposure. The majority of early research in this area focused on factors related to how combat veterans, and Vietnam veterans in particular, responded to war-related trauma (e.g., Egendorf, Kadushin, Laufer, Rothbart, & Sloan, 1981; Figley, 1978; Foy, Sipprelle, Rueger, & Carroll, 1984; Penk et al., 1981; Strange, 1974). Later, researchers began to study the factors that contribute to posttrauma dysfunction in response to other types of traumatic experiences, including natural and technological disasters (e.g., Baum, Cohen, & Hall, 1993; Green, Lindy, Grace, & Leonard, 1992), sexual assault (e.g., Foa, Molnar, & Cashman, 1995; Resnick, Kilpatrick, & Lipovsky, 1991), domestic violence (e.g., Astin, Ogland-Hand, Coleman, &

Foy, 1995; Kemp, Rawlings, & Green, 1991), crime (e.g., Kilpatrick et al., 1989), serious motor vehicle accidents (e.g., Blanchard, Hickling, Taylor, & Loos, 1995), death of a close family member or friend (e.g., Norris, 1992), life-threatening medical conditions (e.g., Green, Epstein, Krupnick, & Rowland, 1997), and acts of terrorism (e.g., Galea et al., 2002; North et al., 1999).

In the remainder of this section, we present a selective review of the literature on psychosocial risk and resilience factors for PTSD, drawing attention to several areas in which additional research is needed. For comprehensive reviews of these factors, the reader is referred to Brewin, Andrews, and Valentine's (2000) recent meta-analysis of this literature, Yehuda's (1999) edited work on the topic, as well as the earlier works of Gibbs (1989) and Green (1994). Although we recognize that there may be a constitutional/hereditary component of PTSD, it is not the focus of this chapter.

As discussed previously, psychosocial risk factors can be categorized into features of the traumatic event itself, preexisting attributes of the trauma victim, and posttrauma circumstances. We summarize research on each of these categories in turn, after which we provide a conceptual integration that highlights the role of available resources and coping strategies as mechanisms through which many of these factors may be implicated.

Features of the Traumatic Event

The largest portion of research has focused on features of the traumatic event itself. Perhaps the most widely studied risk factor is the severity of the traumatic event. There is ample evidence for a dose–response relationship between the severity of the stressor and posttraumatic stress symptomatology (e.g., Fairbank, Keane, & Malloy, 1983; Foy, Carroll, & Donahoe, 1987; March, 1993; Rodriguez, Vande-Kemp, & Foy, 1998). Brewin et al.'s (2000) meta-analysis of risk factors for PTSD revealed a significant effect of trauma severity among both military populations, where the traumatic experience was combat, and civilian populations, in which a variety of other traumatic experiences were represented. In fact, trauma severity was found to be one of the strongest risk factors for PTSD, particularly among military samples.

A number of other important aspects of trauma have been identified as well (Green, 1993), and research has increasingly focused on how these contribute to PTSD. For example, traumas that involve injury have been found to be more highly predictive of PTSD than those that do not (Acierno, Resnick, Kilpatrick, Saunders, & Best, 1999; Green, 1990, 1993; Green, Grace, & Gleser, 1985; March, 1993). Traumas that are more malicious and grotesque are associated with a much greater risk for posttraumatic adjustment problems (e.g., Gallers, Foy, Donahoe, & Goldfarb, 1988; Green et al., 1985; Kessler et al., 1995;

Laufer, Gallops, & Frey-Wouters, 1984). Whether one is actively involved in a traumatic event (as either perpetrator or victim) or merely a witness to a traumatic event affects the likelihood of PTSD (Fontana & Rosenheck, 1999; Green, 1990, 1993; March, 1993); being directly involved in a traumatic event is more likely to lead to a dysfunctional response than being an observer of a traumatic event (Breslau & Davis, 1987; Laufer et al., 1984; Lund, Foy, Sipprelle, & Strachan, 1984). Other aspects of the traumatic experience have been studied as well. For example, King, King, Gudanowski, and Vreven (1995) and Solomon, Mikulincer, and Hobfoll (1987) found that both objective and subjective aspects of war-zone exposure were associated with stress symptomatology. In addition, King et al. (1995) and Litz, King, King, Orsillo, and Friedman (1997) documented the importance of lower-magnitude stressors in association with PTSD symptom severity for Vietnam veterans and Somalia peacekeepers, respectively.

Pretrauma Characteristics

Researchers have increasingly attended to the role of prior trauma exposure in understanding how people respond to subsequent trauma exposure. A number of studies suggest that prior trauma may sensitize an individual to later traumatic experiences, making recovery more difficult (Andrykowski & Cordova, 1998; King, King, Foy, & Gudanowski, 1996; King, King, Foy, Keane, & Fairbank, 1999; Koopman, Classen, & Speigel, 1994; Moran & Britton, 1994; Peretz, Baider, Ever-Hadani, & De-Nour, 1994; van der Kolk & Greenberg, 1987), while other findings indicate that prior trauma might serve to inoculate individuals, thus reducing the impact of the trauma (Bolin, 1985; Burgess & Holmstrom, 1979; Cohen, 1953; Norris & Murrell, 1988; Quarantelli, 1985; Warheit, 1985). Some researchers have suggested that these mixed findings can be reconciled by a consideration of the similarity of previous and current traumatic events; when stressors resemble each other, there may be an inoculation effect, and when they are different, a sensitization effect might result (e.g., Follette, Polusny, Bechtle, & Naugle, 1996; Norris & Murrell, 1988). However, a recent study failed to fully support this hypothesis among a sample of workers at the site of a major air disaster (Dougall, Herberman, Inslicht, Baum, & Delahanty, 2000), finding support for sensitization effects when stressors differ but not for inoculation effects when stressors are similar. Clearly, additional research is needed to better understand the circumstances under which prior trauma sensitizes or inoculates individuals to the effects of subsequent trauma.

Related to the realm of prior traumatic or stressful experiences are family and early childhood variables that may serve as risk or resilience factors for PTSD. Along these lines, researchers have found associations between self-

reports of earlier childhood abuse and PTSD following exposure to subsequent trauma (Andrews, Brewin, Rose, & Kirk, 2000; Bremner, Southwick, Johnson, Yehuda, & Charney, 1993). There is also evidence for the impact of family psychiatric history, as well as early separation from parents, on PTSD (Breslau et al., 1991; Bromet, Sonnega, & Kessler, 1998; Emery, Emery, Sharma, Quiana, & Jassani, 1991). Other adverse childhood experiences associated with PTSD include family instability and poor family functioning (Fontana & Rosenheck, 1994; King et al., 1996; King et al., 1999). Importantly, findings generally appear to suggest that pretrauma family factors may be stronger risk factors for people exposed to military trauma than for civilian populations (Brewin et al., 2000). However, as Brewin et al. (2000) noted, military samples are more likely than civilian samples to include individuals with chronic PTSD, and, thus, it may not be that these risk factors are better predictors of PTSD following military trauma per se but that they are stronger predictors of chronic PTSD than acute stress symptomatology.

Another preexisting factor that may contribute to the development of PTSD following trauma exposure is one's own previous psychiatric history. Individuals who have suffered from various prior or ongoing psychiatric disorders may be more likely to develop other forms of psychopathology (Green, Grace, Lindy, Gleser, & Leonard, 1990; O'Toole, Marshall, Schureck, & Dobson, 1998; Resnick, Kilpatrick, Best, & Kramer, 1992; Schnurr, Friedman, & Rosenberg, 1993). The findings of Schnurr et al. (1993) are particularly compelling, given that the scores derived for preexisting psychopathology were based on data collected before military service rather than collected retrospectively after trauma exposure. Relatedly, early conduct problems as exhibited by antisocial tendencies have been indirectly linked with later PTSD following a traumatic event (King et al., 1996).

Other preexisting demographic and individual factors that have been associated with the development of PTSD include socioeconomic status, education, race, gender, age, and intelligence. In general, findings suggest that lower socioeconomic status, less education, minority racial status, female gender, younger age at trauma, and lower intelligence all serve as risk factors for the development of PTSD following trauma exposure (Brewin et al., 2000). However, these associations are generally quite small. Moreover, the impact of many of these factors may depend on the type of trauma. For example, although findings for socioeconomic status and education were similar across trauma types in Brewin et al.'s (2000) meta-analysis, gender was a risk factor for PTSD following most types of trauma but was not a risk factor for PTSD among military samples. However, gender may be somewhat confounded with combat exposure in many of these studies due to their reliance on samples of

Vietnam veterans, in which most women had only peripheral exposure to combat. A recent study by Wolfe, Erickson, Sharkansky, King, and King (1999) found that gender was a risk factor for PTSD following combat exposure in the Gulf War. Likewise, Brewin et al. (2000) found that younger age served as a risk factor for the development of PTSD following combat exposure but not other forms of trauma. Minority racial status exhibited a weak association with PTSD following combat exposure but was unrelated to PTSD following other types of trauma. Though there was evidence for the impact of low intelligence as a risk factor for PTSD among military samples (e.g., Macklin et al., 1988), no studies on intelligence as a risk factor for PTSD following other types of trauma met the criteria for inclusion in Brewin et al.'s (2000) meta-analysis. Thus, conclusions cannot be drawn regarding the association between intelligence and response to other types of trauma.

Additional research is clearly needed to understand why some demographic/background characteristics serve as risk or resilience factors for some types of trauma and not others. Moreover, as will be discussed in greater detail shortly, research is needed to identify why these factors may serve as risk factors or, more precisely, what the risk mechanisms are through which these demographic/background characteristics have their effect on PTSD.

Posttrauma Recovery Environment

Two primary aspects of the posttrauma recovery environment have received research attention: the social support that is available to trauma victims as they attempt to recover and exposure to additional life stressors. Findings indicate that social support may mediate or moderate the impact of trauma; those with higher levels of social support are likely to suffer less from PTSD than those with lower levels of social support (e.g., Egendorf et al., 1981; Keane, Scott, Chavoya, Lamparski, & Fairbank, 1985; King et al., 1999; Solomon & Mikulincer, 1990; Solomon, Mikulincer, & Avitzur, 1988; Solomon, Mikulincer, & Flum, 1989). Indeed, lack of social support proved to be one of the strongest risk factors for PTSD in Brewin et al.'s (2000) meta-analysis of the literature, and this finding applied equally well to both military and civilian samples.

Additional life stressors after exposure to the index trauma also demonstrate a strong association with posttraumatic distress (Brewin et al., 2000). As King, King, Fairbank, Keane, and Adams (1998), Green (1994), and Resnick et al. (1991) noted, PTSD may be the consequence of a series of highly stressful life events, extending both back into one's personal history (thus, the attention to prior trauma) and forward in time to the present (thus, the attention to additional life stressors).

Risk Mechanisms

Importantly, the risk and resilience factors described earlier may have their impact on PTSD through many different mechanisms (Rutter, 2000a, 2000b). Two issues that deserve special attention are the resources available to an individual exposed to a traumatic event and the coping strategies drawn on as one attempts to deal with a traumatic event and its associated sequelae. Many factors may carry risk through their impact on one's access to resources that can be mobilized for successful adaptation (Hobfall, 1991). For example, many of the demographic and individual factors reviewed previously may have implications for the availability of resources. It may be that individuals who are from lower socioeconomic backgrounds, those who have less education, racial minorities, women, younger individuals, and those who are less intelligent have less access to resources that will facilitate recovery from trauma (see further analysis in the section to follow). Likewise, social support in the period following trauma exposure may signify the availability of both concrete resources, such as financial support, and emotional resources, someone to whom one can turn for empathy and guidance.

Relatedly, factors may carry risk through their impact on coping with the event and its aftermath. For example, the more severe the event, the more threatening the experience may be to an individual's assumption of the world as predictable, controllable, and benevolent, and the more likely may be feelings of "intense fear, helplessness, or horror" (American Psychiatric Association, 1994, p. 428) that challenge one's ability to make sense of and recover from the experience. Coping is an ongoing process and is relevant to many aspects of trauma exposure and recovery: coping with actual trauma-related stressful events and circumstances (e.g., Sharkansky et al., 2000; Sutker, Davis, Uddo, & Ditta, 1995; Suvak, Vogt, Savarese, King, & King, 2002); coping with stress reactions at the time of their occurrence (e.g., Solomon et al., 1989); coping with posttrauma symptomatology (e.g., Fairbank, Hansen, & Fitterling, 1991); and coping with additional postexposure life stressors (e.g., Solomon et al., 1988). Not only is the choice of coping strategies salient, but the effectiveness of coping efforts may have significance for later adaptation. As discussed previously, findings regarding whether prior trauma serves an inoculating or sensitizing role in subsequent response to trauma have been mixed. It may be that this association depends on how successfully one has coped with the prior trauma. Successful coping may lead to positive beliefs about one's ability to cope with adversity (Rutter, 1981), which, in turn, could result in more effective coping with subsequent trauma.

There is also some evidence that certain people may be better able to cope with stressors across the life course. Preexisting hardiness, conceptualized as a

stable intrapersonal resource that facilitates effective coping, may play a protective role in posttrauma adjustment (King et al., 1998; King et al., 1999). In other words, individuals who are hardier to begin with may be more resilient to the effects of trauma. Then, again, there may some traumatic events that overwhelm the coping capabilities of even the hardiest individual. Clearly, additional research attention must be paid to both the risk and resilience factors associated with PTSD and the mechanisms through which these relationships arise.

SEEKING VALID INFERENCE ABOUT RISK AND RESILIENCE: RECOMMENDATIONS TO TRAUMA RESEARCHERS

Understanding how the aforementioned risk and resilience factors influence PTSD necessitates clear thinking about causality and the validity of causal inferences that we draw from empirical work. Although there are variations on the theme of causality, most methodologists impose at least three criteria for valid inference:

1. *Covariation* between the putative causal agent and the effect or outcome: Changes in the cause (e.g., level of postexposure social support) must be accompanied by changes in the outcome (PTSD symptom severity).
2. *Temporal precedence* of the putative cause to the outcome: The causal agent (e.g., the exposure) must precede the outcome (PTSD) in time.
3. *No "third variables"*: All other spurious or alternative explanations for the association (e.g., a prior exposure to an extreme stressor) must be ruled out or deemed improbable.

The gold standard for achieving these criteria is the true experiment in which study participants are randomly assigned to one of a number of researcher-manipulated conditions, the manipulation is performed, and then the outcome is measured. If there are differences between conditions on the outcome, the covariation criterion is met. The design, manipulation followed by assessment, insures temporal precedence. Random assignment reduces the probability that some preexisting factor might explain the findings, as random assignment is intended to eliminate the systematic influence of alternative variables on the outcome.

As noted by King and King (1991) however, research on trauma-related dysfunction can never be truly experimental because the investigator has no control, random or otherwise, to assign individuals to the "treatment"—the

traumatic experience. Furthermore, in the study of etiology, one cannot randomly assign individuals to the types of experiential (e.g., early childhood adversity), personal (e.g., hardiness), or environmental (e.g., a large network of social support structures) attributes that comprise the typical repertoire of risk and resilience factors described in the previous section. Hence, the study of risk and resilience for PTSD mandates an awareness of threats to the validity of causal inference that are endemic to quasi-experimental designs.

Without doubt, the preponderance of PTSD risk and resilience research has been and continues to be passive observational and cross-sectional, usually with retrospective self-reports of features of the traumatic experience itself, as well as retrospective accounts of the victim's pretrauma life and any postexposure events and circumstances that may have occurred up until the time of the investigation. Although such designs may be successful in establishing covariation between a putative risk or resilience factor and PTSD, temporal precedence may be unclear and we are left with ambiguity concerning the direction of cause and effect. King and King (1991) and King et al. (1999) discussed a number of issues that might contribute to such ambiguity. These include demand characteristics of the research setting, researcher-imposed expectancies, response biases such as social desirability or acquiescence, but especially poor recall and associated reconstruction of memory coupled with the tendency for one's contemporary mental state to influence accounts of prior experiences or conditions. For example, one who is highly symptomatic and distraught might judge the extent and degree of trauma exposure to have been quite extreme in some attempt to create balance or congruity in self-conceptions. In like manner, assessments of one's early family life and childhood might be filtered through the lens of current posttraumatic distress, to validate negative appraisals of the self (e.g., Bachman, 1988; Swann, 1983): Does the risk factor (e.g., distressing childhood environment) belong to the causal network responsible for the postexposure condition (PTSD), or does the trauma victim's postexposure condition (PTSD) influence the retrospective report of status on a risk factor (e.g., distressing childhood environment)?

Ambiguity about the direction of cause and effect will continue to be nearly ubiquitous in research on risk and resilience for PTSD. In general, we cannot know in advance who will be exposed, nor can we anticipate and acquire scores on pertinent measures of risk and resilience prior to that exposure. (Some noteworthy exceptions include studies by Card, 1987; Macklin et al., 1998; Schnurr et al., 1993) Yet, careful attention to contemporary methodological theory and techniques can help us confront the challenges to understanding etiology in trauma research. We offer a series of recommendations aimed at enhancing the ability to make valid causal inferences about risk and resilience for PTSD.

1. Recognize the Importance of Precise Risk Terminology and Thoughtfully Delineated Risk Mechanisms

We base this recommendation largely on the thoughtful work of Kraemer and colleagues (e.g., Kraemer et al., 1997, 1999; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001) and Rutter and colleagues (e.g., Rutter, 2000a, 2000b; Rutter & Sroufe, 2000). Several years ago, Kraemer et al. (1997) proposed a taxonomy that provides a highly useful conceptual framework to organize and catalog the many risk and resilience factors identified within PTSD research. Arguing that precise risk terminology is critical for valid causal inference, scientific communication, and appropriate clinical and policy applications, they proposed a decision tree (p. 341) whereby variables appearing to relate to an outcome of interest (*correlates* and therefore potential risk factors for PTSD) can be classified into one of several mutually exclusive categories, each of which carries special meaning with regard to the operation of that factor in the etiological network. When a correlate is observed, a first decision is whether temporal precedence exists (i.e., Is the factor under consideration antecedent to PTSD?). An affirmative response connotes the potential for a *risk* (or *resilience*) *factor.* A negative response would suggest that the correlate is not a risk factor but rather a *concomitant* or *consequence* of PTSD. A good example might be comorbid depression that is observed following exposure to trauma: There is a body of literature (e.g., Breslau, Davis, Peterson, & Schultz, 1997; Erickson, Wolfe, King, King, & Sharkansky, 2001; Skodol et al., 1996) examining the interplay between PTSD and depression and attempting to determine temporal precedence. If we observe that postexposure depressive symptomatology covaries with PTSD symptomatology, evidence that depression is antecedent to the onset of PTSD would make it a likely risk factor candidate (prior psychiatric history). Evidence that depression merely accompanies PTSD but did not actually precede PTSD would place it in the concomitant or consequence category.

Kraemer et al. (1997) elucidated several categories of risk factors. A risk factor that does not vary within an individual over time is termed a "fixed marker." Examples here would be age at exposure to the index trauma, gender, race, ethnicity, and other preexisting, permanent, and unchanging characteristics of individuals that have been shown to be associated with PTSD. A "variable risk factor," conversely, is one that either changes for the individual naturally (e.g., additional life stressors following the index event) or can be manipulated in some way (e.g., the infusion of social support resources into communities following a disaster). Moreover, at the point at which a variable risk factor can be shown to be manipulable (changing social support by providing resources) and, when manipulated, a demonstrated change in the out-

come is achieved (decreased prevalence of PTSD), that risk factor is designated a "causal risk factor."

This taxonomy has implications for those who study trauma. It is important to state, as did Kraemer et al. (1997), that use of the term "causal risk factor" does not imply that the researcher necessarily understands the causal mechanism. In other words, the third variable problem remains. The provision of support resources to victims of a large-scale disaster may result in a reduction in PTSD caseness, but do we really understand the mechanism? It could involve the reduction of additional stressful life events or the enhancement of self-esteem or sense of mastery or some combination of these and other factors. But establishing a risk factor as a causal risk factor gives the researcher a glimpse of possible mechanisms and clues for additional scientific inquiry. Along the same lines, the distinction between a variable risk factor and a causal risk factor may be merely a function of the state of the science at a given point in time. One goal of PTSD research, then, should be to isolate or identify which risk factors are causal risk factors and hence assist in triangulating on the real causal mechanism in the absence of a true experimental design.

In a similar spirit to improve our understanding of risk and resilience in PTSD, we turn to Rutter's (2000a, 2000b) observations regarding *risk indicators* and especially *risk mechanisms*. The former are akin to Kraemer et al.'s (1997) fixed markers, factors that index but do not influence outcomes. The latter appear to be elaborations of the processes by which Kraemer et al.'s causal risk factors are translated into change in outcomes. According to Rutter, an understanding of mechanisms demands that we clearly recognize what risk factors are associated with what variables. In particular, risk factors for exposure to a traumatic event may not be the same factors that explain the mechanism by which exposure leads to PTSD. In some of our own work (e.g., King et al., 1996; King et al., 1999), albeit with cross-sectional and retrospective data, we documented an association between childhood antisocial behavior and combat exposure, but the direct link between childhood antisocial behavior and PTSD was trivial. A prominent intervening variable, or risk mediator, was perceived threat in the war zone. Thus, prior psychopathology (antisocial behavior) operated according to one set of mechanisms in predicting trauma exposure (perhaps via risk taking or thrill seeking) and via another set of mechanisms in predicting PTSD (fear of death or bodily harm).

2. Apply Multivariate Methods to Aid Understanding of Complex Causal Mechanisms

Accumulating knowledge about bivariate associations between each of an abundance of possible risk and resilience factors and a health outcome of interest falls far short of the goal of specifying causal processes, because simple

relationships are not particularly informative of the underlying mechanisms that govern etiology (Kraemer et al., 2001). Causal mechanisms are typically quite complicated, and, especially because we are confined to passive observational quasiexperimental designs, multivariate statistical methods become a critical tool. Recall the third criterion for causality: the no "third variables" requirement. One elaboration of this criterion is in terms of spurious effects by variables outside the observed association that are, in some simple to complex way, responsible for that association and thus may reveal the mechanisms by which the presumptive cause affects the outcome.

Previous analyses using the National Vietnam Veterans Readjustment Study (NVVRS) database offer good examples of how uncovering spurious effects may inform possible causal mechanisms. Consider a *mediational paradigm* (Baron & Kenny, 1986). Using NVVRS data, King et al. (1995) identified and operationalized four dimensions of war-zone experiences (exposure to traditional combat events and circumstances, exposure to atrocities and incidents of extraordinarily abusive violence, perceived threat or fear of death or bodily harm, and exposure to the lower-level discomforts and irritations of the malevolent war-zone environment) and subsequently related these to PTSD symptom severity. Although the bivariate relationship between combat exposure and PTSD symptom severity was quite strong—as it should be—a more revealing and theoretically useful finding was the role of perceived threat as a powerful mediator of this relationship. In the presence of the perceived threat variable, the association between combat exposure and PTSD symptom severity was rendered trivial. Hence, a possible mechanism explaining the association between trauma and PTSD may be the intervening variable of fear of death or bodily harm, with the effect of combat on PTSD being indirect through perceived threat. To some extent, this pattern substantiates the importance of PTSD's Criterion A2. As another illustration with NVVRS data, King et al. (1996) attempted to evaluate a network of premilitary background variables and their indirect and direct associations with PTSD. They pointed to exposure to heavy combat as a possible mechanism for an observed bivariate association between childhood antisocial behavior and involvement in atrocities and abusive violence in Vietnam. That is, the link between childhood antisocial behavior and involvement in atrocities was indirect and via the combat variable; this result might suggest that veterans with prewar behavior problems were not necessarily predisposed to atrocious acts in the war zone, but that the association between these two variables is mediated by or in the context of heavy combat.

In both of the foregoing cases, had not the third variable been introduced into the model, evidence of a potential process by which a risk factor presumably affects another variable would have been lost. Of course, given that the data were cross-sectional and retrospective, the direction of influence is still

problematic, and the specification of the mechanism remains probabilistic and subject to future investigation. Although King et al. (1995) and King et al. (1996) specified and evaluated a mediational model, with perceived threat and combat exposure, respectively, acting as intermediary variables, two other equally plausible models are possible. One alternative is for the third or spurious variable to covary with the antecedent risk factor and have a direct influence on the outcome. A second equally plausible model would have the spurious variable as a "cause" of both the risk factor and outcome. The lesson here is that statistical methods alone are not sufficient to confirm a causal process. Sound statistical methods must be accompanied by solid design and supported by theory.

Multivariate methods are also available to answer questions regarding possible *synergistic or joint effects* among risk and resilience factors, traditionally framed within the context of moderator or interaction analyses (Baron & Kenny, 1986). As a case in point, the literature on coping as a resilience factor in the face of trauma is large and somewhat inconclusive, especially with regard to the value of approach-based or problem-focused coping, engaging in behaviors aimed at active intervention or direct confrontation to resolve the stressful situation. Some studies have found approach-based or problem-focused coping to be beneficial in their negative association with trauma sequelae, whereas others have found rather minimal associations (see Sharkansky et al., 2000; Suvak et al., 2002). Yet, a third variable that might govern the coping-outcome relationship is the general context in which the coping takes place (e.g., Folkman, Schaefer, & Lazarus, 1979; Forsythe & Compas, 1987; Park, Folkman, & Bostrom, 2001): To what extent does the success or failure of the coping strategy depend on features of the stressor that evoke the coping process? One characteristic that comes to mind is a risk factor: the severity of the stressor. In particular, does the coping-outcome relationship vary with the intensity of the exposure, such that understanding the causal mechanism requires both a consideration of the level of expression of coping behaviors and the amount of trauma exposure?

For example, Suvak et al. (2002) hypothesized and demonstrated the synergistic influence of problem-focused coping and degree of trauma exposure in the prediction of long-term educational and occupational achievement among Vietnam veterans. Specifically, the strength of the association between the use of problem-focused coping strategies in the war zone and the outcome varied curvilinearly as a function of the level of combat. Consistent with expectations, at low levels of combat, where there was no need to engage in problem-focused coping, the relationship was negligible. With increasing levels of exposure to combat, and concomitant greater need for coping, the relationship strengthened in the proposed direction: the more problem-focused

coping, the more achievement. And, as exposure to combat became extreme, at a level where problem-focused coping may become less efficacious or even fruitless, the association between problem-focused coping and achievement again weakened. Thus, problem-focused coping may be effective at moderate levels of trauma exposure, but at low levels, it may be unnecessary, and at high levels, it may be irrelevant; hence the curvilinear interaction or quadratic moderator effect. Although Suvak et al.'s outcome was long-term achievement, we have found a similar significant quadratic but inverse problem-focused coping interaction with the outcome of PTSD symptom severity.

Mediator and moderator effects employing continuous outcomes such as PTSD symptom severity are almost always evaluated by means of ordinary least-squares regression-based statistical methods and probably most frequently by some type of multiple regression. (Variations on logistic regression, including polytomous and ordinal logistic regression, with maximum likelihood estimation would apply when the outcome is a categorical variable, such as PTSD diagnosis.) Even more complex systems of relationships can be mapped and assessed through use of a series of simultaneous ordinary least-squares regression equations, to reflect a chain of hypothesized causal mechanisms from more distal risk and resilience factors through more proximal factors and terminating in PTSD. Specification and appraisal of such a network of associations is a *path analysis.* The reader is directed to a comprehensive compilation of applied multiple regression procedures, including an introduction to path analysis, in the new Cohen, Cohen, West, and Aiken (2002) text.

An even more elegant elaboration of multiple regression and path analysis for testing mechanisms of risk and resilience is the class of statistical procedures called *structural equation modeling.* Although structural equation modeling often appears challenging to execute, under certain conditions it can offer distinct advantages over regression-based path analysis. First, when properly implemented, structural equation modeling yields associations among perfectly reliable variables; as a consequence, all parameter estimates are unbiased, a condition that cannot be guaranteed with ordinary least-squares regression and path analysis with measures of varying and less-than-perfect reliabilities. Second, in structural equation modeling, all the information in the data set is used in the estimation of all parameters; thus, parameter estimates are efficient, with standard errors as small as they can be, given the data. Third, structural equation modeling provides estimates of the overall goodness of fit of the multivariate model to the data and allows for the selection of the best in a series of competing models. As in multiple regression analysis, techniques for assessing mediation and moderation are available (see, e.g., Cortina, Chen, & Dunlap, 2001; Jaccard & Wan, 1996; Joreskog & Yang, 1996, for guides to evaluating interaction effects in structural equation modeling).

We recapitulate the warning, however, that elegant statistics, even "causal modeling," do not overcome the fundamental deficiency of a cross-sectional design that might include retrospective accounts of exposure or pretrauma status. In such a research situation, path analysis and especially structural equation modeling can be informative and suggestive of risk mechanisms, but direction of causality is still ambiguous. Bollen (1989), Hayduk (1989, 1996), Loehlin (1998), Kline (1998), and Schumacher and Lomax (1996), among others, provide excellent introductory treatments of structural equation modeling.

3. Consider a Reconceptualization of Outcome as Process

Without doubt, longitudinal research designs are better suited than cross-sectional designs to clarify temporal precedence and thus directionality of associations among key variables. Moreover, longitudinal designs are critical to understanding the course of a posttrauma condition, either naturalistically over time or as a function of intervention or treatment. Initial calls for longitudinal studies of trauma consequences were penned by Green, Lindy, and Grace (1985), Denny, Rabinowitz, and Penk (1987) and Keane, Wolfe, and Taylor (1987), who recommended particular attention to tracking the efficacy of alternative therapeutic interventions. King and King (1991) expanded on these suggestions by endorsing a developmental perspective on trauma research and the incorporation of then-available longitudinal designs and analytic strategies, which could elucidate both temporal precedence and the phenomenology of chronicity or recovery and response to treatment.

Indeed, a number of prominent trauma research teams (Blanchard et al., 1996; Bolton, Litz, Glenn, Orsillo, & Roemer 2002; Foa & Riggs, 1995; Green et al., 1990; Marmar et al., 1999; McFarlane, 1988, 1992; Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992; Shalev, Freedman, et al., 1998; Shalev, Sahar, et al., 1998; Solomon, Benbenishty, & Mikulincer, 1991; Southwick et al., 1995; Ursano, Fullerton, Kao, & Bhartiya, 1995; Wolfe et al., 1999) have increasingly incorporated prospective designs in their work. Most have employed ordinary least-squares regression-based analytical methods in which a respondent's status on prior-measured variables is used to predict standing on subsequent PTSD. The predictor may be the dependent variable assessed at a previous time point, for example, PTSD in the first days following an exposure predicting PTSD at one or more subsequent assessments. The predictor may be one or more risk (e.g., severity of exposure) or resilience (e.g., hardiness) factors measured on an earlier occasion. Or, the predictor may be the presence or absence of, or one or more core features of, a strategic intervention (e.g., early debriefing). In this case, another likely predictor is respondent's status on the outcome prior to treatment (e.g., a baseline PTSD score). The logic here is that

the "partialed" effect of the treatment (in the presence of the baseline score) on the outcome measured on any one of a series of follow-up assessments represents a posttreatment change in status.

Using standard autoregressive statistical models, regression coefficients tell us how a person's standing in relation to the mean of an independent variable assessed on one occasion (say, PTSD at 1 week postexposure) mirrors that person's standing in relation to the mean of the dependent variable assessed on a later occasion (PTSD at 1 month or PTSD at 3 months postexposure). This representation is important but rather static in that there is a prediction of where an individual will end up on the variable of interest (e.g., relatively high or relatively low) but not how that individual got there as a function of time since exposure and various risk and resilience factors. A relevant question is, "Over time, postexposure, whose symptoms will remain nonclinical, whose will diminish, whose will continue to be elevated, and whose might even increase?" Hence, an individual-differences characteristic, an index of an individual's *change in symptoms* over the full time course or across selected time intervals—a statistic describing trajectory, or increasing or decreasing trends in symptom severity, not simply a *score* at some time point—becomes the pertinent dependent variable. Given the shift in outcome from static score to dynamic change, there is likely a concomitant need to readdress risk and resilience factors. This interindividual-differences-in-intraindividual-change perspective was endorsed by Rutter (2000a), who noted that stronger causal inference is possible when change in a risk or resilience factor is accompanied by within-individual change in an outcome over time.

As with developmental and aging research, which uses age or time since birth as a fundamental component of this within-individual change methodology (McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002), research on the sequelae to traumatic events has a start date, the point of exposure, from which the manifestation of an individual's symptoms can be mapped and profiled as a function of time. This distinguishing aspect of trauma-based mental health research makes it ideal for the application of methods designed to explicate interindividual differences in intraindividual change and thus to gain knowledge of what points in time-since-exposure symptom trajectories may be critical to understanding influences on chronicity and recovery and most accommodating to intervention. These modern approaches include accelerated longitudinal or cohort-sequential designs (Bell, 1954; McArdle & Bell, 2000), latent growth curve modeling applied to continuous data (e.g., McArdle & Epstein, 1987; Meredith & Tisak, 1984, 1990; Willett & Sayer, 1996) and to categorical data (Muthen, 1996), random coefficients regression (Raudenbush & Bryk, 2002; McArdle et al., 2002), and dynamic latent difference score analysis (Hamagami & McArdle, 2001; McArdle, 2001; McArdle & Hamagami, 2001).

Willett (1988), Ragosa (1995), and Lawrence and Hancock (1998) provide excellent introductions to the concept of interindividual differences in intraindividual change.

4. Index and Appraise Effect Sizes of Risk and Resilience Factors

In recent years, research in the behavioral, social, public health, and biomedical sciences has been experiencing a paradigm shift with regard to the reporting and interpretation of empirical findings. Reliance on what is known as null-hypothesis-significance-testing logic has been proclaimed an impediment to the advancement of scientific knowledge, and a growing number of prominent methodologists have called for its actual ban in the research literature (e.g., Hunter, 1997; Kirk, 1996, 2003; Schmidt, 1996, Thompson, 2002). Journals in public health and epidemiology have taken a lead in disallowing the reporting of significance tests in published articles, and the American Psychological Association commissioned its Task Force on Statistical Inference to study the issue and make recommendations for reporting strategies in its scientific publications.

The primary criticism of null hypothesis significance testing is that it misrepresents the probability of an incorrect decision. Although it is true that if, indeed, the null hypothesis is correct and there is no effect or association, the probability of a Type 1 error or alpha is an accurate probability of an incorrect decision. But, critics note that under most conditions, the alternative hypothesis is correct and there is some level of effect or association. As a consequence, the error is not in falsely rejecting the null hypothesis but rather in falsely rejecting the alternative hypothesis of an effect or association (where probability equals 1 – power). In other words, reaching a conclusion of nonsignificance under the condition of the alternative hypothesis is incorrect. Associated with this technical argument are several misguided beliefs held by researchers who employ null hypothesis significance testing:

1. That a statistically significant association at a given probability level (an alpha of .05 or .01) means that the probability of replicating the finding in future analyses is 1 – alpha. In actuality, this is false; the probability of replication is the power, which is unrelated to alpha but is related to the true effect size.
2. That the *p* value according to the calculated test statistic is an index of the size and importance of the effect or association. In actuality, the *p* value is a function of the sample size or degrees of freedom and should never be used to interpret how "highly significant" a finding is.

3. That nonsignificance is equivalent to no effect or association. In actuality, nonsignificance does not translate to an effect of zero or no association.

Schmidt (1996) provides an excellent elaboration of these falsely held beliefs and related issues.

In lieu of significance testing, the emerging recommendation is to report effect sizes with accompanying confidence intervals, a practice that is particularly appealing in research on risk and resilience factors for PTSD and other health outcomes. Two arguments are offered in support of this recommendation. First, the reporting of effect sizes for risk and resilience factors allows for a consideration of the relative strengths of the effects and not simply a dichotomous yes–no decision, which might dismiss an effect that is small yet informative. Even a weak effect or an effect with a calculated confidence interval that includes zero can have theoretical and possibly practical meaning. Second, the reporting of effect sizes and confidence intervals encourages a meta-analytic point of view in which the researcher's attention is drawn to the accumulation of findings in an effort to arrive at more precise and encompassing estimates of relative effect size across a collection of studies, perhaps a collection of populations. To date, there have been few meta-analytic studies on trauma and its effects (see those by Kaylor, King, & King, 1987; Rubonis & Bickman, 1991; Weaver & Clum, 1995); Brewin et al.'s (2000) work is especially important in the risk and resilience arena, and the very recent meta-analysis by Ozer, Best, Lipsey, and Weiss (2003) is likewise an extraordinarily valuable resource. More quantitative syntheses of this type are encouraged. There are a good number of texts on effect sizes, confidence intervals, and meta-analytic techniques, including those by Cooper and Hedges (1994), Harlow, Mulaik, and Steiger (1997), Hunter and Schmidt (1990), Lipsey and Wilson (2001), and Oakes (1986). A recent special section in *Educational and Psychological Measurement* contains a series of articles on the computation of effect sizes and their confidence intervals for different statistical procedures, along with suggested computer scripts (e.g., Cumming & Finch, 2001; Fidler & Thompson, 2001; Smithson, 2001).

IMPLICATIONS FOR PREVENTION, TREATMENT, AND POLICY

Historically, clinicians and policymakers have taken a "one-size-fits-all" approach to the provision of programs and services to individuals exposed to traumatic events (Litz, Gray, Bryant, & Adler, 2002). This is unfortunate because not everyone who is exposed to trauma is at equal risk for experiencing posttraumatic symptomatology and response to exposure certainly varies.

Only recently have practitioners and planners begun to attend to the literature on risk and resilience factors for PTSD (Everly, 2000) and to apply these findings to clinical and policy decision making concerning management of risk (Kraemer et al., 1997). Although this trend is encouraging, a lack of understanding about the causal inferences that can be drawn from studies of risk and resilience can considerably reduce the usefulness of this information for prevention, treatment, and policy (Litz et al., 2002), and valuable resources may be squandered on interventions that fail to have the desired impact.

Kraemer et al.'s (1997) risk taxonomy is quite valuable in a service planning and delivery context. Programs that target factors that cannot readily be affirmed as antecedents to the outcome, referred to as concomitants or consequences by Kraemer et al., are unlikely to achieve a reduction in PTSD. Yet given the cross-sectional nature of much of the risk and resilience literature, as well as the fact that concomitants or consequences of PTSD are likely to demonstrate stronger associations with PTSD than variables that meet the criteria for causal risk factors, early intervention practitioners attend to posttraumatic symptoms and functional impairments to the detriment of other factors, such as fixed markers and causal risk factors, that may have more promise for informing intervention efforts (Kraemer et al., 1997).

Risk factors that are considered fixed markers are useful for targeting a population that is most vulnerable and should receive services, especially when assets are limited. They are likewise useful in identifying groups that deserve priority screening. Whereas fixed markers have their primary role in the identification of individuals to target for programs and services, to the extent that the mechanism through which fixed markers have their impact on PTSD can be identified, this information can also be used to inform service delivery. For example, the finding that the association between race or ethnicity and PTSD is mediated through the impact of the former on the availability of resources that can facilitate recovery might inform programs aimed at providing those resources that are found to be critical to recovery.

Causal risk factors are most revealing when a program of prevention or intervention is planned, with of course the intention that manipulation via the program will produce changes in outcome (Kraemer et al., 1997). Rutter (2000a, 2000b) likewise endorsed the planning of prevention and intervention research based on sound knowledge of the processes that underlie causal links between risk factors and outcomes; mechanism-based treatments are not only more likely to succeed but are more etiologically informative. Interestingly, PTSD presents an unusual case for the Kraemer et al. risk taxonomy in that there is more often than not an identifiable time of exposure to an event or circumstance such that many individual characteristics of the victim *at the time of exposure* are fixed markers. Thus, although current age and current marital

status may be variable risk factors—with persons in different age groups demonstrating varying levels of mental distress—age or marital status at time of exposure is a fixed marker.

Of course, the impact of causal risk factors on PTSD may be moderated or mediated by other variables, and knowledge of these associations can inform practice. For example, the previously described finding that some coping strategies are only effective under certain circumstances (e.g., moderate trauma exposure vs. high or low exposure; Suvak et al., 2002) cautions against preventive programs that encourage a particular coping strategy regardless of contextual factors. Relatedly, much of the evidence on the impact of risk and resilience factors on PTSD suggests that these factors operate differently for different trauma populations (Brewin et al., 2000). Therefore, those who deliver services or those responsible for policy decisions about prevention or intervention must be well informed about the repertoire of risk and resilience factors that may operate differentially, depending on context or trauma population. In addition, knowledge regarding indirect associations between risk and resilience factors and PTSD (i.e., mediator effects) can inform prevention and intervention efforts. Some risk factors may demonstrate indirect associations with PTSD or have their effect on PTSD through other factors in the chain of causation.

The recognition that most associations between risk factors and PTSD are quite complex brings us to yet another issue with regard to the literature on risk and resilience for PTSD. Sometimes, what appears to be a simple association between a particular risk factor and PTSD may be complicated by a third variable. Rutter (2000b) offers an excellent example: The simple recognition that parental divorce is associated with negative outcomes for children could lead practitioners and policymakers to conclude that a reduction in divorce is necessary to avoid these negative outcomes. A very different approach would be elicited by the knowledge that the causal mechanism through which divorce affects outcomes is by its indirect effects through parent–child relationships and parenting practices.

In addition, as Kraemer et al. (1997) noted, different features of the time course of a psychiatric entity (time of onset and trends toward recovery, remission, or relapse) may be predicted by different sets of risk and resilience factors, an observation that fits well with our call for a reconceptualization of outcome as process. Thus, as a case in point, interventions need to be geared to the particular stress phenomenon that is being targeted (e.g., chronic PTSD in response to combat-related trauma vs. acute stress symptomatology expressed in the immediate aftermath of an act of terrorism).

Finally, the reporting of effect sizes, or risk factor *potency* (Kraemer et al., 1997, 1999), and the accumulation of effect size statistics over studies are like-

wise meaningful for clinical and policy decisions. Without this information, it is impossible to differentiate between those associations that represent clinically meaningful relationships and those that are so small as to be practically meaningless. Risk factors that have little potency may be of negligible value to clinical, policy, and research application (Kraemer et al., 1997), and limited resources may be misspent when intervention efforts are geared toward risk factors that have little practical impact on PTSD. Of course, the importance of a particular effect size may be up for debate, and researchers may determine that a fairly small effect is of practical significance. What is key here is that practitioners move beyond simply attending to statistical significance to a recognition that effects differ in magnitude and that those of a higher magnitude may be better candidates for intervention.

In conclusion, practitioners are urged to undertake prevention, treatment, and policy efforts with knowledge about the validity of causal inference and its implications for the conclusions that can be drawn from the existing literature on risk and resilience for PTSD. More generally, the recognition that causal mechanisms are quite complicated cautions against universal or rigidly applied interventions. Interventions should be informed by knowledge pertaining to how factors operate in the etiological network, as well as the recognition that these factors may operate differently for different trauma populations, or even subpopulations within trauma populations (e.g., women versus men; minority versus nonminority). Attention to these issues can result in more effective prevention, treatment, and policy efforts and ultimately can lead to a reduction in the negative consequences of exposure to traumatic events.

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